Childs Nerv Syst (2006) 22: 562–566
DOI 10.1007/s00381-006-0060-4

BRIEF COMMUNICATION

Andrea Klein
Bettina Balmer
Ulrike Brehmer
Thierry A. G. M. Huisman
Eugen Boltshauser

Facial nerve palsy—an unusual complication after evacuation of a subdural haematoma or hygroma in children

Received: 15 July 2005
Revised: 23 September 2005
Published online: 14 March 2006

A. Klein (✉) · E. Boltshauser
Department of Neurology,
University Children's Hospital Zurich,
Steinwiesstrasse 75,
8032 Zurich, Switzerland
e-mail: andrea.klein@kispi.unizh.ch
Tel.: +41-44-2667330
Fax: +41-44-2667163

B. Balmer
Department of Pediatric Surgery,
University Children's Hospital Zurich,
Steinwiesstrasse 75,
Zurich, Switzerland

U. Brehmer · T. A. G. M. Huisman
Department of Diagnostic Imaging,
University Children's Hospital Zurich,
Steinwiesstrasse 75,
Zurich, Switzerland

Abstract *Objective:* This paper reports and discusses on the possible etiology of postoperative contralateral facial nerve palsy after uneventful evacuation of a subdural haematoma or hygroma after mild head trauma in two children with pre-existing middle cranial fossa subarachnoid cysts. *Results:* Two 14- and 15-year-old boys had prolonged headaches after mild head injuries. CT showed a right-sided middle cranial fossa arachnoid cyst in each patient. In one patient, an ipsilateral subdural haematoma was identified, and in the other, bilateral hygromas were identified. Exacerbation of symptoms required emergency evacuation of the subdural haematoma in the first child, and bilateral external drainage of the hygroma in the other child. In both children the late postoperative period was complicated by peripheral facial nerve palsies contralateral to the arachnoid cyst. *Conclusion:* Facial nerve palsy may be a complication of hygroma or haematoma drainage. The etiology is not clear; traction of the facial nerve due to displacement of the brainstem may be the most likely explanation.

Keywords Facial nerve palsy · Arachnoid cyst · Brain stem displacement · Subdural haematoma

Introduction

Facial nerve palsy after the evacuation of subdural haematomas or hygromas has not been reported before. We present two patients recently seen at our hospital that both developed facial nerve palsy after the drainage of trauma-induced hygroma in one patient, and haematoma in the other. Each child had a pre-existing middle cranial fossa arachnoid cyst. Arachnoid cysts are congenital intra-arachnoid cerebrospinal fluid collections and account for about 1% of all intracranial space-occupying lesions in children. They are mostly asymptomatic, most often located in the middle cranial fossa and are known to predispose for subdural haematoma and hygroma after mild head injuries. Usually, evacuation of the haematoma and/or hygroma will relieve the neurologic symptoms, the arachnoid cysts usually do not have to be fenestrated [1, 2]. The goal of our report is to describe the clinical and

imaging findings in these children and to discuss the possible pathogenesis of the facial nerve palsies.

Patients

Case 1 A previously healthy 15-year-old boy presented with a prolonged headache and vomiting after a mild head injury. A cranial computed tomography (CT) scan performed 2 weeks after the trauma showed a right-sided arachnoid cyst in the middle cranial fossa without evidence of post-traumatic lesions. Initially, there was slight improvement of symptoms but the recurrent headaches and vomiting persisted. Seven weeks after the trauma, his condition acutely worsened with signs of elevated intracranial pressure and herniation. A repeat CT showed a significant right-sided subdural haematoma that compressed the adjacent intra- and extracerebral CSF spaces. In addition, a midline shift was observed (Fig. 1). An emergency trepanation with evacuation of the supratentorial subdural haematoma and a subdural drainage were performed. The early postoperative period was uncomplicated, but on the fifth postoperative day a left-sided peripheral facial nerve palsy was noted. The postoperative magnetic resonance imaging (MRI) on days 1 and 12 after surgery did not show any pathology along the course of the left facial nerve or within the brainstem (Fig. 2a–c). The brainstem, however, appeared slightly displaced to the right side; the left parapontine cistern was somewhat wider compared to the right side (Fig. 2c). MRI showed discrete ischaemic cortical lesions in the right lateral occipito-temporal gyrus and the left cingulate gyrus, most probably resulting from a transient subfalcial herniation in the acute phase of the subdural haematoma. The facial palsy was still moderate (House Brackman lll) after 12 months follow-up.

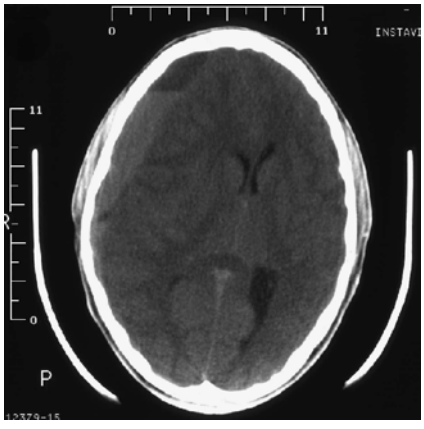

Fig. 1 Axial CT. Subacute, partially hyperdense subdural haematoma with blood-serum sedimentation level along the right cerebral hemisphere. A resulting moderate midline shift in combination with a compression of the right ventricle is seen

Case 2 A 14-year-old boy had a mild head injury in a car accident without loss of consciousness. In the following weeks he developed vertigo, headaches and vomiting. Cranial CT performed 2 weeks after the trauma revealed a right-sided middle cranial fossa arachnoid cyst and bilateral hygromas. On follow-up MRI 5 days later, the hygromas had increased in size (15 mm wide). There was no sign of bleeding, the ventricles were normally wide and there were no signs of a cerebral displacement or herniation. The brainstem was unremarkable (Fig. 3a,b). On follow-up, diplopia occurred. Consequently, a bilateral external drainage of the hygromas was performed 3 weeks after the initial trauma. The patient's headache resolved promptly and his diplopia improved. However, on the third postoperative day a left-sided peripheral facial nerve palsy was noted. MRI did not reveal any pathology along the course of the facial nerve, but the same signs of a brainstem displacement as in the first patient were seen (Fig. 3c). Again, the left parapontine cistern was slightly wider as compared to that on the right side. Oral steroids were given (2 mg/kg/day for 5 days). The facial nerve palsy gradually improved, with a discreet residual asymmetry 2 months later. Because of recurrent hygromas after disconnecting the external drainage, a subduro-peritoneal shunt was placed. The shunt had to be removed after 6 weeks because of a subacute shunt infection. There was no recurrence of hygroma.

Discussion

Arachnoid cysts are developmental anomalies that occur most often in the middle cranial fossa (50%), more often in males and more frequently on the left side [2, 3]. Increase in size is rare [4]; spontaneous disappearance has been described [5, 6]. Most cysts are asymptomatic [7]. There are reports of one-sided macrocrania, headaches and epilepsy. In children with posterior fossa cysts, unspecific symptoms, including ataxia, vertigo and facial nerve palsy [8, 9], have been described. Well-known complications of middle cranial fossa cysts include subdural, intracystic [6] and, rarely, extradural bleedings [1, 10]. Cyst ruptures and the formation of hygromas are rare and have been described in several case reports [10–12]. Subdural haematomas may also occur [6]. In mild head trauma, chronic subdural haemorrhages or hygromas most often occur ipsilateral to the arachnoid cyst, sometimes they can be bilateral, and rarely they occur in the contralateral side of the arachnoid cyst [2, 6]. The bleeding risk of non-operated arachnoid cysts has been estimated to be low (0.04% per year) [2]. Spontaneous cyst rupture and disappearance is rare [2, 13].

Neurosurgical treatment remains controversial. Evacuation of haematoma or hygroma without cyst fenestration has been shown to be sufficient in most cases [2, 6]. Spontaneous reduction of the cyst after evacuation has

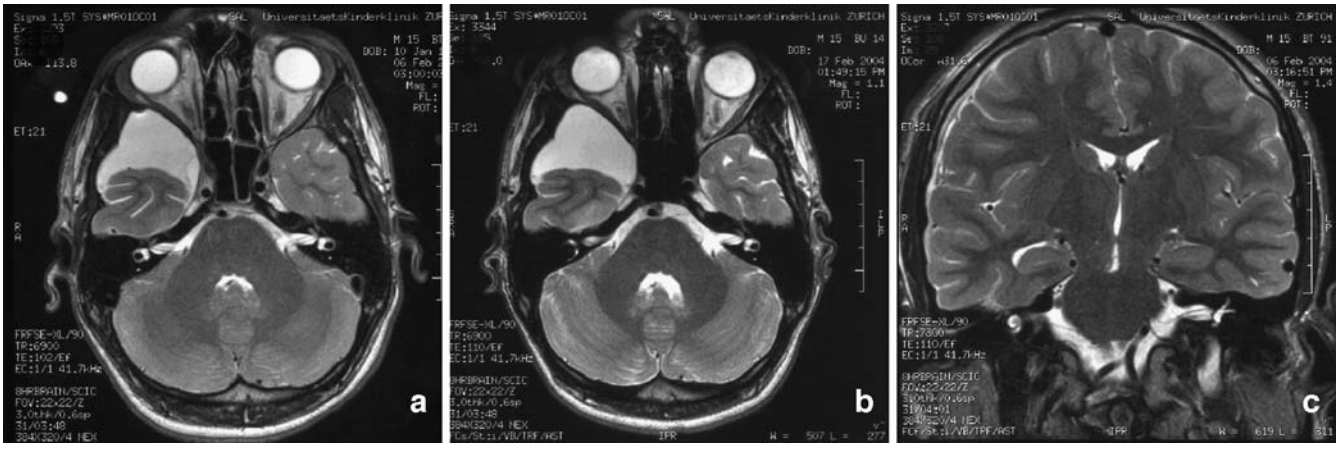

Fig. 2 Serial axial (**a**, **b**) and coronal (**c**) T2-weighted fast spin-echo MRI. The early and follow-up axial images show an intact arachnoid cyst within the left middle cranial fossa in combination with a moderate temporal lobe hypoplasia (**a**, **b**) The pons is slightly displaced to the right side with asymmetry of the parapontine cistern, left > right (**a**, **b**, **c**). No signal abnormalities are seen within the brainstem

been observed [6]. In cases of recurrent hygromas, subduro-peritoneal shunting may be necessary [2]. Although there are many reports of evacuated and drained haematomas or hygromas with or without cyst fenestration, we did not find a report on complicative facial nerve palsy after evacuation.

Both our patients had very similar underlying pathologies and clinical courses; therefore, coincidence is very unlikely. The facial nerve seems to be sensitive to pressure changes or traction. In the two cases of facial palsy in patients with posterior fossa arachnoid cysts, traction of the facial nerve has been proposed as a possible mechanism [8]. Another report describing bilateral abducent and facial nerve palsies following fourth ventricle shunting also hypothesised that facial nerve palsy could result from a brainstem displacement that results in a stretching of the trunk of the facial nerve between the brain stem and the internal acoustic canal [14]. The imaging findings in both our patients of a slight displacement of the brain stem to the contralateral side of the facial nerve palsy would be compatible with the hypothesis of a functional deficit resulting form a stretching of the facial nerve. As the facial nerve palsy was contralateral to the cyst, direct nerve damage during the surgical intervention is excluded. Extracranial preauricular facial nerve compression during the intervention is also not possible because of the way the patients were positioned during the operation.

In patients with pseudotumour cerebri (benign intracranial hypertension), facial palsy occurs rarely. Presentation may be bilateral and is often combined with other cranial nerve dysfunction [15, 16]. However, other cranial nerves, such as the abducens, trochlear and oculomotor nerve, are much more frequently involved. The presumed causative mechanism for the encountered cranial nerve palsies in

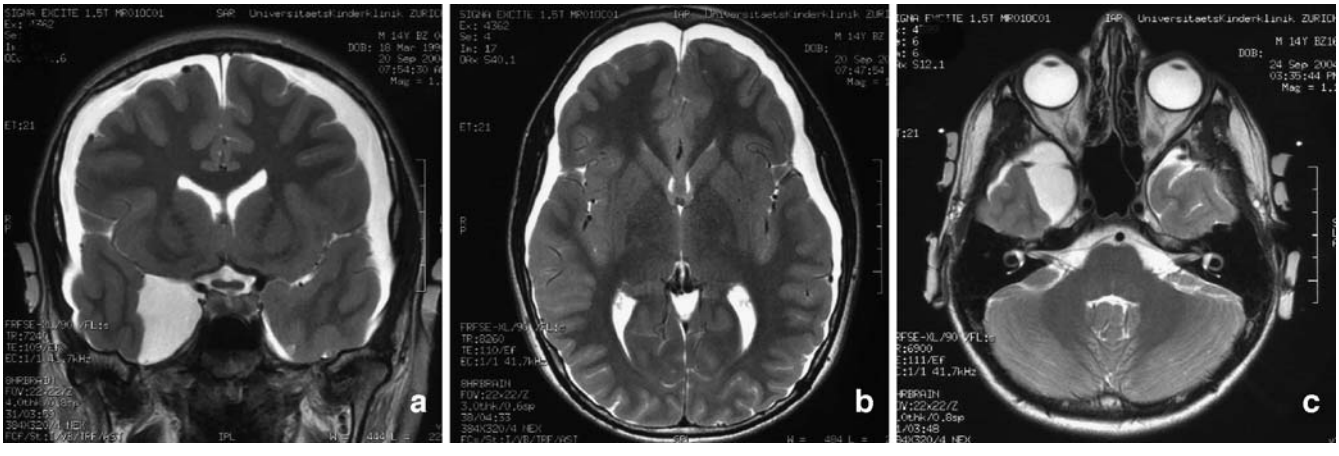

Fig. 3 Serial axial (**a**, **c**) and coronal (**b**) T2-weighted fast spin-echo MRI. Initial axial (**a**) and coronal (**b**) MRIs show bilateral T2-CSF isointense subdural hygromas along both cerebral hemispheres, as well as a small right middle cranial fossa arachnoid cyst. Follow-up axial MRIs (**c**) showed a discrete wider left parapontine cistern compared to the contralateral side. No brainstem pathology is seen

pseudotumour cerebri is again traction to the extra-axial segments of the nerves by the elevated intracranial pressure.

In patients with thrombosis of the dural sinuses, isolated multiple cranial nerve palsies are described, often with involvement of the facial nerve. Venous congestion with oedema was proposed to be the reason for nerve dysfunction [17]. In a case report of a patient with extended cerebral venous thrombosis with facial palsy, a partial conduction block proximal to the entrance of the nerve into the facial canal was demonstrated. As pathomechanism, the authors proposed a rise in intraluminal venous pressure that led to a venous blood–brain barrier dysfunction and therefore an impairment of the saltatory current flow that explains the reversible conduction block. The observed facial palsies were reversible after recanalisation of the thrombosed sinuses [18]. This mechanism seems very unlikely in our patients because of prolonged symptoms and the lack of recovery in the first case.

Delayed facial nerve palsy is a well-known complication after acoustic neurinoma resection. Impaired microcirculation, oedema and viral reactivation of herpes simplex virus and varizella zoster virus have been discussed as pathomechanisms [19–21]. In the case of proven viral reactivation, gadolinium enhancement of the facial nerve was present [21]. The lack of facial nerve enhancement as seen in our second case excludes an infectious etiology of the facial nerve palsy with a high degree of confidence.

The delay of the facial nerve palsy in our patients is not understood. A similar delay is observed in patients with acoustic neurinoma resection and in patients with delayed visual loss after surgery of large retrochiasmatic tumours [22, 23]. In the latter cases, a change of microvascularisation of the optic nerve is discussed as a possible pathomechanism.

In conclusion: (1) Our cases show that haematomas or hygromas should be excluded in children with arachnoid cysts who suffer from persisting symptoms after mild head injury. (2) Facial nerve palsy may be a complication of hygroma or haematoma drainage. The mechanism is not yet clear, but the encountered persisting displacement of the brainstem to the contralateral side of the facial nerve palsy indicates that resulting traction of the facial nerve may be causative.

References

1. Galassi E, Gaist G, Giuliani G, Pozzati E (1988) Arachnoid cysts of the middle cranial fossa: experience with 77 cases treated surgically. Acta Neurochir Suppl 42:201–204
2. Parsch CS, Krauss J, Hofmann E, Meixensberger J, Roosen K (1997) Arachnoid cysts associated with subdural hematomas and hygromas: analysis of 16 cases, long-term follow-up, and review of the literature. Neurosurgery 40:483–490
3. Wester K (1999) Peculiarities of intracranial arachnoid cysts: location, sidedness, and sex distribution in 126 consecutive patients. Neurosurgery 45:775–779
4. Artico M, Cervoni L, Salvati M, Fiorenza F, Caruso R (1995) Supratentorial arachnoid cysts: clinical and therapeutic remarks on 46 cases. Acta Neurochir (Wien) 132:75–78
5. Inoue T, Matsushima T, Tashima S, Fukui M, Hasuo K (1987) Spontaneous disappearance of a middle fossa arachnoid cyst associated with subdural hematoma. Surg Neurol 28:447–450
6. Mori K, Yamamoto T, Horinaka N, Maeda M (2002) Arachnoid cyst is a risk factor for chronic subdural hematoma in juveniles: twelve cases of chronic subdural hematoma associated with arachnoid cyst. Neurotrauma 19:1017–1027
7. Gosalakkal JA (2002) Intracranial arachnoid cysts in children: a review of pathogenesis, clinical features, and management. Pediatr Neurol 26:93–98
8. Pirotte B, Morelli D, Alessi G, Lubansu A, Verheulpen D, Fricx C, David P, Brotchi J (2005) Facial nerve palsy in posterior fossa arachnoid cysts: report of two cases. Childs Nerv Syst 21:587-590
9. Boltshauser E, Martin F, Altermatt S (2002) Outcome in children with space-occupying posterior fossa arachnoid cysts. Neuropediatrics 33:118–121
10. Galassi E, Tognetti F, Pozzati E, Frank F (1986) Extradural hematoma complicating middle fossa arachnoid cyst. Childs Nerv Syst 2:306–308
11. Donaldson JW, Edwards-Brown M, Luerssen TG (2000) Arachnoid cyst rupture with concurrent subdural hygroma. Pediatr Neurosurg 32:137–139
12. Gelabert-Gonzalez M, Fernandez-Villa J, Cutrin-Prieto J, Garcia Allut A, Martinez-Rumbo R (2002) Arachnoid cyst rupture with subdural hygroma: report of three cases and literature review. Childs Nerv Syst 18:609–613
13. Poirrier AL, Ngosso-Tetanye I, Mouchamps M, Misson JP (2004) Spontaneous arachnoid cyst rupture in a previously asymptomatic child: a case report. Eur J Paediatr Neurol 8:247–251
14. Spennato P, O'Brien DF, Fraher JP, Mallucci CL (2005) Bilateral abducent and facial nerve palsies following fourth ventricle shunting: two case reports. Childs Nerv Syst 21:309–316
15. Capobianco DJ, Brazis PW, Cheshire WP (1997) Idiopathic intracranial hypertension and seventh nerve palsy. Headache 37:286–288
16. Santos S, Lopez Del Val LJ, Mostacero E, Tejero C, Casadevall T, Morales F, Pascual LF (2001) Pseudotumor cerebral: analisis de nuestra casuistica y revision de la literatura. Rev Neurol 33:1106–1111
17. Kuehnen J, Schwartz A, Neff W, Hennerici M (1998) Cranial nerve syndrome in thrombosis of the transverse/sigmoid sinuses. Brain 121:381–388

18. Straub J, Magistris MR, Delavelle J, Landis T (2000) Facial palsy in cerebral venous thrombosis: transcranial stimulation and pathophysiological considerations. Stroke 31:1766–1769
19. Gianoli GJ (2002) Viral titers and delayed facial palsy after acoustic neuroma surgery. Otolaryngol Head Neck Surg 127:427–431
20. Scheller C, Strauss C, Fahlbusch R, Romstock J (2004) Delayed facial nerve paresis following acoustic neuroma resection and postoperative vasoactive treatment. Zentralbl Neurochir 65:103–107
21. Ohata K, Nunta-aree S, Morino M, Tsuyuguchi N, Haque M, Inoue Y, Ogura H, Hakuba A (1998) Aetiology of delayed facial palsy after vestibular schwannoma surgery: clinical data and hypothesis. Acta Neurochir (Wien) 140:913–917
22. Menke E, Osarovsky E, Reitner A, Matula C (2002) Funktionelle Befunde vor und nach Eingriffen am optochiasmalen System. Wien Klin Wochenschr 114:33–37
23. Pierre-Kahn A, Sainte-Rose C, Renier D (1994) Surgical approach to children with craniopharyngiomas and severely impaired vision: special considerations. Pediatr Neurosurg 21(Suppl 1):50–56